



UNITED STATES NAVY

MEDICAL NEWS LETTER

Editor - Captain L. B. Marshall, MC, USN (RET)

Vol. 24

Friday, 24 December 1954

No. 12

TABLE OF CONTENTS

Residency Training Policy.....	2
Hodgkin's Disease and Amyloidopathia	3
Treatment of Sickle Cell Anemia	4
Natural History of Korean Vivax Malaria.....	7
Blood Transfusion Accidents.....	8
Osteomalacia in New York	10
Pulmonary Kaolinosis.....	12
Atypical Facial Pain	13
Fluorides in Water Supplies	15
Dental Cavities and General Health	17
The Effect of Complete Mouth Rehabilitation.....	18
Urinary Uropepsin	19
Solitary Circumscribed Lesions of the Lung	21
Disappearing Bones.....	22
"Honors"	24
Training Course in Special Weapons, Isotopes, and Military Medicine.	24
Notice of VD Postgraduate Course	25
Chemical Warfare Defense Training Film	26
The Navy Mutual Aid Association.....	27
From the Note Book	27
Board Certifications	29
Narcotic Drugs, Purchase of (BuMed Notice 6710)	31
Submission of NavMed Forms L (BuMed Notice 6630).....	31
Number of Outpatient Visits (BuMed Notice 6320)	32
<u>AVIATION MEDICINE DIVISION</u>	
USS Bennington Casualties.....	32
Carbon Monoxide Determination..	34
Pressure Demand Oxygen Mask ..	35
Physiology Training Manual	36
Pressure Breathing	36
Founders Group	37
Aero Medical Association.....	38
SF-88's	39

Policy

The U.S. Navy Medical News Letter is basically an official Medical Department publication inviting the attention of officers of the Medical Department of the Regular Navy and Naval Reserve to timely up-to-date items of official and professional interest relative to medicine, dentistry, and allied sciences. The amount of information used is only that necessary to inform adequately officers of the Medical Department of the existence and source of such information. The items used are neither intended to be nor susceptible to use by any officer as a substitute for any item or article in its original form. All readers of the News Letter are urged to obtain the original of those items of particular interest to the individual.

* * * * *

Notice

Due to the critical shortage of medical officers, the Chief, Bureau of Medicine and Surgery, has recommended, and the Chief of Naval Personnel has concurred, that Reserve medical officers now on active duty who desire to submit requests for extension of their active duty for a period of three months or more will be given favorable consideration.

* * * * *

Residency Training Policy for Reserve Medical Officers on Active Duty

The response by Reserve medical officers to the Residency Training Program for Reserve officers, as provided in BuMed Instruction 1520.7, has been most gratifying. There are several vacancies remaining in the following residency programs: Pathology, Orthopedic Surgery, Obstetrics and Gynecology, Pediatrics, and Urology. A very limited number of billets are still available in Otolaryngology, Anesthesiology, and Ophthalmology. While applications for training in the above specialties should be for one year at a time, it is expected that in most instances officers who participate in this program will be permitted to complete their required training without interruption. Every effort will be made to accomplish this insofar as service needs will permit.

Reserve medical officers on active or inactive duty, who have completed their obligated active duty imposed by the Universal Military Training and Service Act, as amended, are eligible for participation in this program. Reserve officers on inactive duty must request return to active duty in order to be assigned to such training.

Eligible and interested medical officers should make applications to the Bureau of Medicine and Surgery, via the chain of command. Letters of application should contain an agreement to volunteer for the period of residency training requested and to remain on active duty in the Navy for a period of one year following completion of training, for each year of training received.

From time to time the list of medical specialties in which shortages exist will be published in the Medical News Letter. (ProfDiv, BuMed)

* * * * *

Hodgkin's Disease and Amyloidopathia

In 1840 Rokitansky first described a pathological condition which he called "lardaceous disease." Later Budd designated this condition as "waxy disease." The name "amyloidosis" was given by Virchow in 1854, and is still used although without justification. From the pathological point of view, there is no degeneration of cells but rather a deposit of a substance in the intercellular spaces of the basal layer of the capillary and arteriolar media. "Amyloidopathia" or "amyloidoforme infiltration" is, therefore, thought to be a more suitable name.

Amyloidopathia may be primary, i. e., without obvious cause, or secondary to other conditions. These latter include: (1) suppurative chronic foci in the body, such as tuberculosis, osteomyelitis, bronchiectasis, abscess, et cetera; (2) non-suppurative chronic processes, such as syphilis, chronic arthritis, multiple myeloma, malaria, and Hodgkin's disease. Clinically, the primary cases differ from the secondary group in the absence of any signs of a pathological condition which might lead to amyloidosis.

Amyloidopathia, complicating Hodgkin's disease, has been described in a small number of cases. It is comparatively easy to establish a diagnosis of amyloidopathia in a case of Hodgkin's disease. Attention should be focused on edema, hepatosplenomegaly, albuminuria, absence of erythrocytes and granular casts in the urine sediment, low blood pressure, normal eyegrounds, hypoalbuminemia, low albumin-globulin ratio and hypercholesterolemia, with uremia, a high pH, high levels of creatinin and uric acid in the blood, and renal failure. The Congo-red test is of decisive importance. According to the authors' observations, this may usually be considered positive for amyloidosis if over 40% of the dye disappears from the blood during the first hour. In the first case a disappearance of 13% was estimated while the amyloidosis was obviously already present. It was believed, therefore, that the test should be carried out twice with an interval of some months. If the percentage in this "comparative Congo-red test" increases, it would be considered justifiable to make a diagnosis of amyloidopathia, apart from the absolute figures.

Prognosis may be established on the basis of the difference in the two tests.

What is the relation between amyloidopathia and Hodgkin's disease? Is it only a coincidence or a relation of cause and effect? Apparently the relationship is not coincidental. In the first place, there is no increase in the incidence of amyloidopathia in other diseases, such as tuberculosis or suppurations. In the second place, in none of the reported cases has there been additional disease present which could have been the cause of amyloidopathia. It can thus be concluded that it was related in some way to Hodgkin's disease. Several possibilities can be contemplated:

1. The x-ray treatment may have caused the amyloidopathia. In other cases, however, larger doses have been administered without this effect. Also, it has been shown experimentally that neither single massive nor small and repeated doses can produce amyloidosis--they can cause only degeneration.
2. The presence of a virus or biochemical change could be responsible for both the Hodgkin's disease and the amyloidosis. This possibility has been much discussed, but no final conclusion is so far established.
3. A third possibility is that the histologic changes in the kidneys in Hodgkin's disease result in the production of amyloid substance. Jackson and Parker, however, showed that of 259 cases of Hodgkin's disease, 13 had specific infiltration into the kidney without signs of amyloidosis, and similar findings were recorded in 8 of 32 cases of Hodgkin's sarcoma.

What is the practical value of a diagnosis of amyloidopathia? Grayzel and Jacobi in 1938, and Trasoff et al., in 1944, Lush in 1948, and Skelton in 1951, reported improvement following injection of liver extract in cases of amyloidopathia and chronic rheumatic arthritis. In a second case there were indications of the influence of this extract. In view of this, the establishment of an early diagnosis might be of some importance because treatment may possibly retard the deposition of amyloid substance and the appearance of uremia. The second case suggests that the protein level and the albumin-globulin ratio in the blood, systematically checked every three to six months, may afford an early indication of impending amyloidosis. (Radiology, Nov., 1954; A. Hochman, M.D., P. Czerniak, M.D., Rothschild Hadassah University Hospital, Jerusalem, Israel)

* * * * *

Treatment of Sickle Cell Anemia

Surveys of Negroes in the United States have revealed the incidence of the symptomless sickle cell trait to be about 9% and the incidence of sickle cell anemia to be about 2.25 per thousand.

It now seems well established that the sickling phenomenon is due to a gene which produces sickle cell anemia when it is present in the

homozygous state and sickle cell trait when it occurs in the heterozygous state. The erythrocytes in both conditions are abnormal and assume the characteristic "sickle shape" when the hemoglobin is in the reduced form. This characteristic of the cells has been shown to be due to hemoglobin that is different from the normal electrophoretically. Erythrocytes in patients with sickle cell anemia contain only the abnormal sickle cell hemoglobin, while those of persons with the sickle cell trait contain a mixture of the abnormal sickle cell hemoglobin and normal hemoglobin. A hemolytic type of anemia occurs because hematopoiesis, although increased, is not able to compensate for the excessive erythrocyte destruction. In many patients hemoglobin and erythrocyte levels remain essentially the same over a period of years because the life span of the red cells and the maximum compensatory effort that the bone marrow is able to make remain unchanged. On occasions this balance is upset, and the anemia rapidly becomes much more severe, either as a result of increased hemolysis or as a consequence of the development of a temporary aplastic state in the bone marrow. Aside from the sickling property of the erythrocytes and the presence of anemia, probably the most characteristic feature of sickle cell anemia is the occurrence of sudden attacks of pain or other localized manifestations in almost any part of the body. These episodes appear to be explained by the capillary engorgement and areas of infarction that are found in the various organs at autopsy.

Correction of the anemia and the reduction of the in vivo sickling and blood viscosity have been attempted by numerous methods. Opinions on therapy sometimes differ as to the measures that should be employed and the indications for their use. This difference probably is due in large part to the difficulty of evaluating nonspecific treatment in a disease whose clinical course varies as widely as does that of sickle cell anemia.

Transfusion is one of the oldest and most widely used forms of treatment. The dangers associated with its use, such as hemolytic reaction, viral hepatitis, and hemosiderosis, are well known. Transfusions should be used only when there is a definite indication. Multiple transfusions will correct the anemia because the normal erythrocytes transfused into a patient with sickle cell anemia survive for the normal time. Transfusions also produce other desirable results that are not always considered. As the sickle cell erythrocytes survive only a few weeks, multiple transfusions not only correct the anemia but also markedly reduce the number and concentration of sickle cell erythrocytes in the circulation, because the transfusions depress hematopoiesis and the production of sickle cells in the recipient. As a result, the patient's blood can be rendered essentially normal for a period of at least several weeks by the use of multiple transfusions. Quicker results may be obtained by a modified exchange transfusion method which is more suitable for children than for adults.

The place of splenectomy in the treatment of this disease remains controversial. There is agreement that splenectomy is not a procedure

that should be applied to all patients with sickle cell anemia because the operation does not cure the hemolytic process and does not affect the sickling phenomenon. Furthermore, virtual "autosplenectomy" has been found at autopsy in some patients who suffered from a severe form of the disease before death. Consideration of the procedure is limited to children and adults whose spleens are moderately or greatly enlarged.

The anemia of sickle cell anemia rarely responds to anything but transfusions, and as a result the average patient with sickle cell anemia receives no treatment most of the time. The patient remains somewhat handicapped but not ill. The hemoglobin and red cell count remain stable, sometimes for a period of years. Treatment of the anemia with vitamin B₁₂ and liver extract are ineffective, as would be expected. Iron is contraindicated because the anemia may promote iron absorption and increase iron in the body tissues without any increase in iron utilization. Other measures that are useful in some hematological conditions have been tried in sickle cell anemia. The use of corticotropin (ACTH) in one patient was followed by a rise in erythrocytes to normal, a decrease in sickling, and a return of osmotic and mechanical fragility to normal. Subsequent reports have suggested a possible use of cortisone and corticotropin in crisis and in the treatment of leg ulcers. The precipitation of crisis by corticotropin and cortisone has also been noted. Because of these conflicting results, further studies of these drugs are necessary before they can be evaluated. The somewhat surprising report that cobalt chloride produced hematologic improvement in four patients with sickle cell anemia also needs further investigation.

Some of the clinical manifestations of the disease, such as congestive heart failure, cerebral vascular accident, urinary tract hemorrhage, and priapism, are not peculiar to sickle cell anemia and are treated as they are when they occur in other circumstances. Other features of sickle cell anemia may require special measures. In some patients leg ulcers heal with the local application of antibiotics, but the more troublesome ulcers require bed rest and transfusions. Anesthesia, surgery, and pregnancy carry greater risk for the patient with sickle cell anemia than for the normal person and require special consideration. As the increased risk is probably related to the anemia and to vascular accidents secondary to the sickling of the erythrocytes in the circulation, multiple transfusions prior to anesthesia or delivery seem worthy of trial because both the anemia and the sicklemia can be virtually abolished within a week or two by this means.

The occurrence of a severe episode of "crisis" is a serious development for the patient with sickle cell anemia. Although the patients usually recover from these episodes, death from sickle cell anemia in childhood and early adult life usually comes in an episode of "crisis" or during some intercurrent infection. At such times the patient may appear critically ill, with severe anemia, shock, and abdominal pain. If severe anemia is

present, oxygen and transfusions of whole blood or packed cells are indicated. The oxygen should not be continued over a long period, because it has been shown to depress marrow function. Episodes of "crisis" often follow infection or fever, and for this reason infection should be searched for in various parts of the body and should be treated if possible. Surgical consultation and sound judgment are often necessary in patients with abdominal pain, for patients with sickle cell anemia may have the same surgical emergencies as other patients, and it should not be assumed that the pains are necessarily manifestations of the sickle cell disease. Relief of the abdominal pain and back pain may be very difficult. Mild sedation and analgesics may suffice for the milder back and abdominal pains, but even narcotics may fail to relieve the excruciating pain that some patients experience. Recently, benzazoline (Priscoline) was reported to give dramatic relief of pain in the cases of seven children with sickle cell anemia, and this drug warrants further study.

At present the treatment of sickle cell anemia is neither specific nor curative and leaves much to be desired. It is hoped that continued progress in knowledge of the disease will lead to more satisfactory therapy. In the meantime, judicious use of methods now available can contribute to the comfort and survival of the unfortunate persons who are afflicted with this disease. (Arch. Int. Med., Nov., 1954; B.S. Leavell, M.D., School of Medicine, University of Virginia, Charlottesville)

* * * * *

Natural History of Korean Vivax Malaria

One of the important medical problems encountered during the military activities on the Korean peninsula was a very high incidence of vivax malaria. Prior to 1950, Korean malaria had attracted relatively little attention. Since 1950 considerable information has developed relative to the natural history of the disease in man as well as the response of this disease to treatment. Hankey and associates reported on the relapse pattern in naturally acquired Korean malaria as it appeared in over 1500 returning American military personnel, and Burnett reported on an outbreak of Korean malaria in 34 Camp Fire girls in California who had been infected by a carrier returned from Korea.

A strain of Korean vivax malaria was transferred by mosquitoes into 6 nonimmune white men and 4 of these subjects were followed to a point well beyond the apparent spontaneous termination of the disease. It appears from these cases that Korean vivax malaria is similar in its natural history to the other temperate zone malarias (Korteweg, James and Shure, Schuffner and associates, and Coatney and colleagues), and follows the observations already made on naturally acquired Korean vivax

malaria (Hankey and associates, and Burnetti). It is also apparent from these 6 cases that the season of the year has little direct effect on the relapse pattern once infection is produced, nor does the occurrence or lack of occurrence of an early primary attack depend on a variation of the intensity of the sporozoite inoculum.

The most important observation to be drawn from the disease produced in these 6 volunteers was that the malaria of Korea is similar in relapse pattern to the malaria of other temperate zones. Temperate zone malarias usually have an early primary attack followed by a long latent interval, and then repeated late relapses.

Because the primary interest in these cases was directed at the natural history of this disease, each relapse was treated with quinine or chloroquine. These drugs apparently do not disturb the tissue phases of vivax malaria. Each attack was also treated rapidly and vigorously in order to keep immunity at a minimum. It was hoped thereby to reduce the effect of immunity in limiting the course of the disease.

It is apparent that, after quinine treatment, relapses occurred with great rapidity and regularity. The average latent period from the end of quinine therapy to the next parasitemia was 10 days. Chloroquine, by virtue of its long persistence in the body, appeared to abort one or two subsequent attacks, after which the previous periodicity would appear.

The behavior of the 6 heavily-infected patients, in whom the exact date of infection was known, confirms the impression gathered from studies of malaria acquired naturally in the field, namely, that the disease terminates spontaneously before 18 months. Four cases were followed from 9 to 18 months beyond this terminal period, and no evidence of continuing activity was obtained. About 3 years after infection 500 ml. of whole blood from each of 3 patients was subinoculated into 3 nonimmune volunteers by direct transfusion. The volunteers so inoculated remained negative for more than 30 days. (J. Lab. & Clin. Med., Nov., 1954; J. Arnold, M.D., A.S. Alving, M.D., R.S. Hockwald, M.D., C.B. Clayman, M.D., R.J. Dern, M.D., and E. Beutler, M.D., University of Chicago)

* * * * *

Blood Transfusion Accidents

Blood transfusion has become such an important aspect of surgical and medical treatment that the procedure should be made as free from danger and as fool-proof as possible. This report discusses some of the preventable, untoward reactions from blood transfusion and what can be done to avoid them in order to protect the patient from danger and the physician and hospital from unpleasant and costly lawsuits. The fundamental

basis of a safe blood transfusion is accurate blood grouping and cross matching tests. The physician giving the transfusion is completely dependent on the laboratory because mere inspection of the bottle of blood will not help him to determine whether it has been correctly grouped and labeled or whether the patient's blood has been correctly grouped and cross matched. Once blood has been incorrectly grouped and cross matched and released from the laboratory, a hemolytic transfusion reaction is inevitable in certain cases, therefore, these tests should be carried out only by fully qualified persons. The field of blood grouping has become highly specialized and adequate pretransfusion tests require familiarity not only with the A-B-O groups but also with the Rh-Hr types and such special cross matching methods as the conglutination test, the antiglobulin test, and the proteolytic enzyme test. These tests must be carried out by trained technicians who specialize in this field and not by untrained interns or physicians who have only an incidental interest in the subject. The problem posed by emergency transfusions arising at night, over week ends, or on holidays, is best solved by the use of specially trained medical students who are glad to have the opportunity to do this type of work as an aid in meeting their expense at school. An alternative plan is to have the blood grouping tests done at central blood banks which operate at all hours. When grouping and cross matching tests are entrusted to untrained persons, dangerous transfusion reactions are bound to occur.

The surest safeguard against hemolytic transfusion reactions, caused by blood group incompatibility, is to have the pretransfusion grouping and cross matching tests performed by specially trained persons. For a properly functioning transfusion service, qualified persons must always be available. Aside from errors in blood grouping, the greatest dangers to the patient are from clerical errors, ignorance, inexperience, or sheer carelessness. Every person concerned in a blood transfusion is responsible for reading all labels to be certain that blood is transfused only to the patient for whom it is intended. The use of "universal" donor blood should be reserved for dire emergencies only, and in such cases, the transfusionist must be prepared to vindicate his action should a reaction occur. Cross matching tests must be done routinely by the conglutination and the agglutination methods. Moreover, for recipients who may have been sensitized by previous transfusions or pregnancies, the antiglobulin cross-matching test must be carried out in order to detect incompatibilities with respect to blood factors such as Kell, Duffy, and Kidd. Transfusion of contaminated blood and transmission of homologous serum jaundice are two of the calculated risks of blood transfusion. The danger of transfusing contaminated blood can be minimized by limiting the period of storage to 14 days, and by microscopically examining a stained smear of the blood from the bottle immediately before the transfusion. There is no reliable test to detect carriers of the hepatitis virus. The danger of hepatitis from pooled plasma can be reduced by storing the plasma in liquid form at room temperature for 6 months or longer before use, or better, by avoiding pooling

and using only group-specific plasma. Because blood transfusion has many associated hazards, physicians should not prescribe this type of treatment unless it is definitely indicated and essential for the patient's recovery. (J. A. M. A., Dec. 4, 1954; A. S. Wiener, Jewish Hospital of Brooklyn, N. Y.)

* * * * *

Osteomalacia in New York

Osteomalacia, the adult counterpart of rickets, is generally considered a rarity in the western world, because avitaminosis D, the commonest cause of the disease, is thought to occur only in regions where exposure to sunshine is scarce and dietary deficiency and malnutrition are common.

In this part of the world osteomalacia is practically limited to conditions where vitamin D is not absorbed from the intestine. Malabsorption of fat-soluble vitamin D occurs in the chronic fatty diarrhea which is present in the sprue syndrome, in chronic biliary obstruction, and in long-standing obstruction of the pancreatic ducts. In the sprue syndrome, the absorptive powers of the small intestine are impaired; in pancreatic disease, the absence of lipase prevents the hydrolysis of neutral fats which is necessary for fat absorption. In chronic biliary obstruction, the bile acids--important factors for the absorption of fats--cannot reach the lumen of the small intestine. In one patient sclerodermatous changes of the intestinal wall apparently prevented the absorption of fat and fat-soluble substances, among them vitamin D. Patients with long-standing steatorrhea are usually debilitated, and because they do not expose themselves regularly to sunshine no vitamin D can be formed in the skin. The combination of malabsorption of vitamin D and lack of formation of this vitamin results in avitaminosis D.

Several cases seen at various hospitals in New York City illustrate the occurrence of osteomalacia in association with disturbances of the absorption of fats and fat-soluble substances from the gastrointestinal tract.

Osteomalacia is a metabolic disease of bone. In this condition insufficient amounts of calcium and/or phosphorus are available. This prevents normal calcification of bone matrix, and in the long run, demineralization results.

The exact mechanisms by which calcium and phosphorus are precipitated from the dissolved ions circulating in the extracellular fluids are not yet known. It is obvious, however, that calcium and phosphorus will precipitate only when the solubility product of these two ions is exceeded.

Regardless of the mechanisms which bring about the precipitation of the calcium bone salts, as soon as the body is seriously depleted of either calcium or phosphorus, the solubility product of the two ions cannot be exceeded. Then precipitation of calcium phosphate does not take place and newly formed osteoid does not calcify. Therefore, conditions which cause a negative calcium or phosphorus balance ultimately lead to a failure of the second stage of bone formation. If such a situation occurs in the growing child, then rickets develops.

In the adult, lack of absorption of calcium and phosphorus from the intestine leads to osteomalacia. Although in the adult the epiphyses are closed, there is still new formation of bone going on throughout the entire span of life. The adult bone is in a dynamic equilibrium. Continually a small amount of bone is resorbed and the same amount of bone is laid down in a process which proceeds slowly from day to day. In compact bone, this resorption and new formation take place along the cement lines of the haversian systems. In cancellous bone, it occurs at the edges of the trabeculae. In the adult, as in the child, calcification of newly laid down osteoid suffers if the body stores of calcium have been depleted. The lamellae and trabeculae of preexisting bone become surrounded by a broad layer of non-calcified bone matrix which histologically presents itself as eosinophilic-staining osteoid. The histologic finding of broad osteoid zones is a striking feature both in rickets and in osteomalacia.

After a state of calcium depletion has existed for a while, a considerable proportion of the skeleton comes to consist of osteoid, and the bones are weakened for lack of mineral material. Bone pains appear, and roentgen examination of the skeletal system reveals generalized demineralization and possibly pathologic fractures. Often, roentgenograms disclose the presence of small fissures in the cortex of the long bones, consisting of proliferation of osteoid which remains uncalcified. These pseudo-fractures are designated as Looser's zones or Milkman's fractures. The serum calcium and phosphorus are low, reflecting the depletion of the calcium and phosphorus stores of the organism. For the same reason, practically no calcium is excreted in the urine. The finding of any abnormally low serum calcium and/or inorganic phosphorus is an essential feature of rickets and osteomalacia, without which the diagnosis cannot be made. Four cases of osteomalacia observed in New York City are reported. All cases suffered from fatty diarrhea. In 2 cases the steatorrhea was due to chronic jaundice, in 1 case to scleroderma, and in 1 case to long-standing ileostomy.

The pathogenesis of osteomalacia, occurring in cases of chronic steatorrhea, is discussed and the importance of the loss of fat-soluble vitamin D in the fatty stools is emphasized. Treatment should consist of parenteral vitamin D administration and radiation with ultraviolet

light. (Ann. Int. Med., Nov., 1954; I. Snapper, M.D., and I. Feder, M.D., Brooklyn, N. Y., R. Seely, M.D., and S. Falk, M.D., New York City)

* * * * *

Pulmonary Kaolinosis

Although the term kaolinosis occurs in a widely circulated medical dictionary, the name is apparently not used in the few available published reports of pneumoconiosis associated with exposure to the inhalation of dust of clay. At the present time there are only two proved varieties of chronic pulmonary disease developing from the inhalation of particulate matter in the atmospheres of industrial plants or workings--silicosis and asbestosis. The present report sets up a definite disease that may be disabling and may reach an advanced or even fatal stage. While the condition described has certain features that are not clearly distinguishable from silicosis, it has other features which warrant the special designation, at least until some particular part of kaolin may be shown to be the disease-causing factor.

The material, from which the dust concerned in this study came, was apparently derived by open mining and was processed in an industrial plant for distribution and use in various industries. The exposure apparently occurred in such a processing plant. Previously, before the recognition of such hazards or possible hazards, doubtless some phases of the processing created very dusty atmospheres. According to the authors' observations, those conditions seem to have been largely corrected. This study shows, however, that wherever workers are exposed sufficiently to inhalation of air heavily laden with particles of kaolin, some may develop chronic disease of the lungs as a consequence.

An additional factor that may need to be taken into account is from the addition of chemicals in the processing of the clay. Soda ash, trisodium phosphate, and sodium pyrophosphate are sometimes used.

While roentgenograms in two cases observed apparently do not furnish features upon which definite differentiation between silicosis and kaolinosis may be made, two observations of possible significance may be made from them. The first is, that while the same features may show in cases of silicosis, the massiveness of the involvement of the upper parts of the lungs in kaolinosis is remarkable. Kaolin dust is composed of very fine particles and is comparatively light, possibly having a better opportunity to reach the upper lung areas than heavier dust composed of larger particles. Secondly, although much pulmonary air space is obliterated, emphysema is prominent in the open areas.

The bluish color created by the deposits in the lungs was a distinctive feature. While the reactions causing the development of blue color

in clay materials are apparently not clearly understood, it is believed that, because of their absorptive qualities, clay crystals may develop a blue reaction under some conditions when mixed with organic amino compounds. Whether this reaction and the consequent development of blue color would occur under all circumstances of deposition of kaolin in the lungs cannot be ascertained except from extensive observation, but it should constitute a distinctive feature when it is found, identifying the nature and source of the deposit even if it were not otherwise known.

From the microscopic descriptions it will be noted that while nodular fibrosis with massive whorled collagenous deposits, as in silicosis, composed the more bulky changes, fibrosis of the alveolar walls and thickening and emphysema were conspicuous. This explains the prominence of respiratory difficulties, at least in the one case in which information was available.

Also to be noted, are the associated vascular lesions. These consisted of extensive obliterative arteritis in which visible particles of foreign material were scattered through the vessel walls, even in the fibrous tissue which occupied the lumina of some arterial branches, and in the first case, thrombosis of the main branches of the pulmonary artery.

Sufficient exposure to inhalation of dust with at least some kaolin deposits will cause, in some individuals, a chronic fibrous disease of the lungs that may be disabling and even prove to be fatal. The term kaolinosis is, therefore, established as signifying a definite disease state beyond merely the location of kaolin in the lungs. (Am. J. Path., Nov-Dec., 1954; K.M. Lynch, M.D. and F.A. McIver, M.D., Medical College of South Carolina, Charleston, S.C.)

* * * * *

Atypical Facial Pain

Many writers have drawn attention to the problem of atypical facial neuralgia, and there are as many names for this condition as there are treatments for its relief. It has been called atypical migraine, trigeminal migraine, sympathetic neuralgia, sphenopalatine neuralgia, and histamine cephalgia. Unfortunately, owing to the difficulties in understanding this condition, and in finding a satisfactory treatment for this atypical facial pain, many cases have been thought to be "neurotic."

The clinical symptoms of 40 patients were studied. In all of these cases the diagnosis had been made by a recognition of the characteristics of the pain, usually in association with other symptoms. The condition was found to be more common in females than in males, 24 women (60%), and 16 men (40%). The onset of the pain was most common in the fourth and fifth decades (57%). The presenting symptom in all was pain in the head or face. The pain was usually described as burning or boring, and

varying in intensity, at times being so severe as to make some sufferers feel suicidal. The distribution of pain was over a wide area and was not confined to one sensory nerve. It was usually in and behind the eye, in the cheek, nose, and jaw, and supra-orbitally spreading backward over the head, in the occiput and neck, and sometimes into the preauricular and mastoid areas. In a very few patients the gums and palate were also affected. The pain could occur in any of these sites alone or in any combination, or in all simultaneously, but the basic pattern remained the same for each patient. Nearly all had pain in the orbit and the eye, and in the occiput and neck. Each patient was asked to draw on a prepared diagram the distribution of his pain, indicating the areas of greatest intensity by depth of shading. The pain was usually unilateral but in 2 cases was bilateral and of equal intensity on both sides; in 7 cases there was slight pain on the opposite side during an acute attack.

Twenty-two patients complained of constant discomfort in the affected area with attacks of exacerbation; 18 had intermittent pain with complete freedom between attacks, and in 2 of these the pain was always nocturnal and in another 2 was almost completely nocturnal. The acute attacks of pain would last from several hours to 2 or 3 days. In addition to the cranio-facial pain, painful sensations were felt in the neck during an attack, and in 1 patient involved the retro-sternal region; another patient had slight objective sensory loss in the hand of the affected side.

The occurrence of various other symptoms, attributable to disturbance of the sympathetic nervous system, was of value in diagnosis. Many patients had flushing of the affected side of the face during an attack. Localized sweating, lacrimation, a sensation of nasal congestion and pupillary changes localized to the affected side; occasionally ptosis and a Horner's syndrome were found. Of these symptoms lacrimation was by far the most common.

Glaser has drawn attention to these symptoms and included nausea and vomiting. Some patients in the study showed these symptoms along with severe pain, but on the whole this was more rare than indicated by Glaser. In this connection, it is important to note that vomiting did not relieve the pain in any way.

In this series of cases no constant precipitating factors were observed, but 16 patients noticed that movement of the neck could exacerbate the pain and that extension relieved it. For example, some patients noticed that the pain came on when the neck was flexed in performing such tasks as sewing, reading, floor-polishing, and ironing. One patient related the pain to cold. However, apart from the position of the neck no precipitating factor was noticed by the patients. The pain in no way resembled trigeminal neuralgia. There were no trigger zones and the pain was not precipitated by eating, talking, washing, et cetera. The authors consider that atypical facial neuralgia is related to the sympathetic nervous system in the neck and to the release of a metabolite which is histamine.

The basis of treatment has been to break a vicious circle on either the efferent or the afferent side. Any interruption on the afferent side, such as section of the great auricular nerve, will probably prove disappointing. By far the most useful therapy was immobilization of the neck, although some cases needed different treatment.

The authors state that their argument includes many factors not adequately explained or proved and that they hope to continue their study. However, they believe that many patients can be relieved by trial of the various treatments outlined in this article. (Lancet, Nov., 20, 1954; A.M.G. Campbell, Bristol Royal Hospital, J.K. Lloyd, Bristol United Hospitals, Bristol, England)

* * * * *

Fluorides in Water Supplies

Although dental diseases rarely cause death, their costs in money, time, and pain are enormous. In the United States during the year ending 1 July 1953, a total of 1.6 billion dollars is reported to have been spent for various forms of dental care, including the repair and replacement of teeth damaged or lost through tooth decay and diseases of the gums. This amount equals 15.7% of the total national health bill of 10.2 billion dollars. Unquestionably this expenditure for dental treatment far exceeds the financial cost of any other single health problem among American citizens. Still these figures do not reveal the true magnitude of dental disease in the United States. In the same survey, it was estimated that only 17% of persons in families with incomes less than \$2000 sought dental treatment; even in families with incomes of \$7500 or more, only 56% had any dental care during the year.

In the past few decades, many precise and esthetically pleasing procedures have been developed and effectively used for the restoration of tissues destroyed by dental disease. Despite all these procedures, there are indications that tooth decay is occurring more rapidly now than as recently as two decades ago.

On the basis of present knowledge about the prevention of tooth decay, only one proposal appears to have a tangible potentiality for appreciably reducing the amount of tooth decay on a nation-wide basis. This proposal is to increase the fluoride content of public water supplies.

The following five questions are discussed in the article: (1) Is a beneficial nontoxic procedure to increase the fluoride content of public water supplies feasible? (2) What are the fluorides and where are they found? (3) Is the ingestion of fluorides beneficial? (4) Is the prolonged ingestion of fluorides at low levels toxic? (5) Can fluorides be used safely and effectively in any other way?

From the data presented in discussion of the preceding five questions, it was concluded that (1) bodies routinely metabolize small amounts of inorganic fluoride that are present in all foods; (2) the ingestion of an optimum amount of inorganic fluorides during tooth development results in a 50% lower incidence of tooth decay through adolescence and adult life; (3) the consumption of this amount of fluorides does not result in any toxic manifestations even after long periods; (4) the fluorides can be as accurately introduced in waterworks as by nature; and (5) there is presently no method of supplying fluoride that is as safe and effective as fluoridation.

It is doubtful that any other public health procedure has been tested with as many patients under as many different controlled circumstances for as long periods. Nature has provided dozens of communities from the north to the south of this country with every conceivable level of water-borne fluorides, from the most minute traces to 8 p.p.m. or more. Thus there has been available for study a wealth of epidemiologic material that could not possibly have been collected in a humanly planned survey. The data from these communities all points toward the rational use of fluorides at a level of about 1 p.p.m. as the only known way to reduce tooth decay in urban populations.

Opponents of fluoridation frequently talk about the toxic manifestations that occur at very high levels of fluoride ingestion as if they likewise occur when water contains only the recommended amount. No scientist would disagree with the statement that fluorides are poisonous at very high levels of ingestion. What is not pointed out by the opponents is the now well-established fact that there are no toxic manifestations at the recommended levels of fluoride ingestion. Like so many substances that are essential to well-being, the fluorides have a broad spectrum of physiologic influences, ranging from deficiency signs at suboptimal levels of consumption to a definitely beneficial phase at optimal levels, thence to a toxic phase at a much higher rate of intake. It is of the greatest importance to recognize the difference between these three stages and, particularly, the difference between the second and the third. Where the safety factor between the toxic and the beneficial levels is as large as for fluorides, no reason exists for withholding the benefits of the active agent from the public.

The only foreseeable tangible danger with respect to the fluoridation of public water supplies involves the possibility that its dramatic benefits may lessen the willingness of the public to support dental research or decrease the zeal of independent investigators to continue their research concerning tooth decay. Even when all urban areas with water systems have incorporated optimum amounts of fluorides sufficiently long to attain full effectiveness, tooth decay will not be fully prevented among city-dwellers. In addition, the inhabitants of rural areas, which comprise approximately 45% of the population, will still be untouched. There can be no longer any doubt that the fluoridation of public water supplies represents the first major step in the prevention of tooth decay, yet it is

not an end or complete answer in itself. The search must be increased for other ways to combat tooth decay and other dental diseases. None of these potential methods are likely to replace fluoridation, but rather they may be expected to supplement its action or increase its effectiveness. (Scient. Month., Oct., 1954; K. S. Quisenberry, Agricultural Research Service, U. S. Dept. of Agriculture)

* * * * *

Dental Cavities and General Health

Applications for school, employment, insurance, and even personal communications typically contain an assessment of the state of a person's general health. The condition of health is described in broad terms, such as good, fair, or poor, dependent mainly on the number or severity of bodily disorders a person has. Type or types of disorders leading to this designation are not necessarily considered. Furthermore, in all such descriptions the oral cavity and its pathology are almost always neglected. This may be largely due to the fact that dental disease and its treatment is a highly developed specialty which appears to be peripheral to the rest of medicine. A person who has numerous dental cavities filled during a year's time is rarely thought to be in "poor" health. Therefore, the possibility of a relationship between dental pathology and other bodily dysfunction on this level has never been seriously considered. A study was undertaken to ascertain whether such a relationship exists.

The 8-month cumulative medical histories of a sample of Naval Aviation Cadets were examined. This group comprised a total of 1080 subjects. The name, serial number, complaint, diagnosis, treatment, and disposition of the case were tabulated for each man. It was found that five or more dispensary visits were made by approximately 15% of the total population. Assuming frequency of medical complaints as a health index, these 178 subjects could be assumed to represent the "poorest" on a general health evaluation.

The complete dental records, including roentgenographs of this criterion group, were then collected where possible and DMF (decayed, missing, or filled teeth) ratings made from these records. The DMF rating represents a subject's total past and present dental caries experience. It is compiled by scoring 1 point for each surface of a tooth containing caries or a filling and 3 points for each missing tooth. The resultant mean DMF score of the criterion group was then compared with the mean Naval Aviation Cadet DMF rating formerly established.

Of the 178 subjects in the criterion group, complete dental records were found on 121. The other 57 records were unavailable because the cadets had dropped from the program. The mean DMF established on

an independent sample of 1019 unselected Naval Aviation Cadets is 27.22 with a standard deviation of 15.55. The mean DMF of the criterion group is 31.09 with a standard deviation of 14.76. The t-test for the difference between these means yielded a value of 2.60, which is significant at the .01 level of confidence. It should be noted that this difference was established on a population that can be considered above average in general health, having been screened by a rigorous physical examination prior to admission.

In recent years, dentistry, as well as other branches of medical science, has postulated that certain chronic illnesses affect dental conditions, and conversely. This study, therefore, provides an interesting relevancy. It presents empirical evidence of a relationship between general health, as measured by frequency of medical complaints, and oral pathology measured by a decayed, missing, and filled-teeth rating. This finding demonstrates that dental caries, as well as other somatic complaints, should be considered in any appraisal that attaches a general term, such as good, fair, or poor, to a person's condition of health. (Science, Nov., 1954; J.H. Manhold, LT DC USN, and C.E. Izard, Naval School of Aviation Medicine, Pensacola, Fla.)

* * * * *

The Effect of Complete Mouth Rehabilitation on Oral Lactobacilli Counts

The presence of Lactobacilli has been widely used as an index of dental caries activity. Caries-active subjects usually demonstrate lactobacilli counts of at least 10,000 colonies per ml. of saliva, where caries-inactive individuals seldom exhibit counts above 2000 colonies and often manifest zero counts. This study was performed to determine the effect of complete mouth rehabilitation on oral lactobacilli.

Forty-four naval recruits, ages 17 to 21, at the Great Lakes Naval Training Center were utilized for this project. All carious teeth were to be restored and necessary extractions performed.

The evidence indicated that lactobacilli counts drop considerably when carious lesions are eliminated by restorations with silver amalgam, silicate cement or by extraction. This reduction occurred to a lesser degree in incompletely rehabilitated mouths.

Control of this study was difficult to maintain in many aspects. Due to illness, discharge, official orders, et cetera, many of the patients could not be followed for the period of 15 weeks.

The cases were diagnosed and restorations fabricated at a recruit dental clinic by several operators. The only way it was possible to check whether all carious lesions had been properly diagnosed and subsequently restored was the information noted on Navy Form 603. The charts of 5

incompletely rehabilitated cases showed unfilled lesions. A question may now arise as to why all of the 39 completely rehabilitated cases did not uniformly exhibit zero postoperative counts even though the forms showed that all lesions had been treated. As it was impossible for the authors to recheck the patients along with their pre- and postoperative radiographs, it is not known whether all carious lesions were diagnosed accurately or properly restored. Perhaps an enamel lesion was inadvertently overlooked and, therefore, not recorded, or possibly a carious pit or fissure was not diagnosed.

Many factors may account for a reduction of lactobacilli in rehabilitated mouths. Perhaps the oligodynamic action of amalgam and/or the bacteriostatic effect of silicate restorations played a role in the reduction. But more likely, the elimination of food retention sites and lactobacilli infected carious areas are chiefly responsible for their disappearance. Reduced sugar intake might be considered a factor. However, the recruits were not limited at any time in their carbohydrate consumption; if anything, they consumed more than usual amounts of fermentable material during the period of study. Forty essentially untreated individuals failed to show similar reductions in lactobacilli counts over a three-month period. Other investigations in this laboratory have shown consistently high lactobacilli counts during comparable training periods.

The tendency of lactobacilli counts to drop after mouth rehabilitation should be an important consideration when such counts are used in the evaluation of caries control agents. Those members of a study who receive dental restorations simultaneously with the application of an anti-caries therapeutic procedure, may have their lactobacilli pictures influenced by both factors.

This study will continue with a more careful selection of patients who will be followed over a longer period. More attention will be given to the accuracy of clinical and radiographic diagnosis of carious lesions, and to the extent and quality of rehabilitation performed. (Research Facility, Dental Dept., Great Lakes; NM 008 013.04.11, Oct., 1954)

* * * * *

Urinary Uropepsin

Measurement of the excretion of pepsinogen in the urine (uropepsin) has been recommended as a quantitative test for gastric secretory function by a number of investigators. It has been applied clinically to the study of peptic ulcer, pernicious anemia, and gastric cancer, and has proved to be of considerable value in measuring the gastric response to ACTH stimulation and conditions of stress. The purpose of this presentation is to evaluate the clinical use of uropepsin excretion in a variety of gastro-intestinal and endocrine diseases, as well as in the

postoperative stomach in which gastric aspiration presents technical difficulties.

The measurement of uropepsin excretion appears to be a useful adjunct to the evaluation of a number of gastrointestinal and endocrine diseases. A high level of uropepsin invariably signifies gastric hypersecretion and is highly suggestive of peptic ulcer. Values above 4000 units were found in 95% of all patients with duodenal ulcer, regardless of the activity of the disease, and were not encountered in 120 normal persons. A uropepsin output of greater than 4000 units, however, may also be encountered in gastric ulcer, ulcerating cancer, adrenal and pituitary hyperactivity, patients receiving ACTH or cortisone, and patients under stress, such as that after surgery, burns, and severe pain.

Abnormally low levels of uropepsin output--below 1000 units--have been encountered only in patients with gastric cancer, pernicious anemia, Addison's disease, myxedema, and total gastrectomy. They were not encountered in any normal subject or in the patients with duodenal ulcer, regardless of age, and were seen in only 1 of 68 patients with benign gastric ulcer.

In the presence of an ulcerating gastric lesion, a uropepsin excretion of 1500 units or less indicates a high probability of cancer, since only 4.5% of 68 patients with benign gastric ulcer excreted less than 1500 units. A urinary excretion of over 4000 units favors, but does not prove, a benign lesion, since 66% of patients with gastric ulcer had higher levels in contrast to 14% with cancer.

A low uropepsin per se would probably not be effective as a screening test for gastric cancer because only 61% of patients with cancer excrete less than 1500 units. It may be useful, however, in conjunction with other screening technics for gastric carcinoma.

Uropepsin excretion appears to be of clinical value in differentiating duodenal ulcer from esophageal varices in patients with massive hemorrhage from the gastrointestinal tract. There is a distinct difference in the uropepsin excretion in the two groups in that patients with portal cirrhosis and esophageal varices excrete exceedingly low levels of uropepsin, with a mean of 945 units compared with 8812 units in patients bleeding from duodenal ulcer. In every case of bleeding from duodenal ulcer, the uropepsin values were greater than 3500 units, whereas all patients with varices or cirrhosis of the liver demonstrated a low uropepsin--below 2125 units--with most levels below 1500 units.

Another clinical use of uropepsin determination is in evaluation of patients who have undergone subtotal gastric resection, in whom gastric analysis is inherently difficult and often unreliable. In many cases the uropepsin determinations suggest actively secreting gastric tissue in patients presenting absent or low free acid on gastric aspiration. This low or absent gastric acidity may be attributed in part to intestinal reflux of biliary secretions.

The finding of a uropepsin value higher than 4000 units in a patient with a previous subtotal gastrectomy, whose symptoms have recurred, is highly suggestive of a marginal ulcer. The close relation found between the increase in uropepsin excretion and the urinary corticoids during hormone therapy and accompanying states of hyperadrenocortical and hypoadrenocortical function suggests the possibility of utilizing the uropepsin determination as an index of adrenal responsiveness. A positive correlation has been reported between uropepsin and urinary 17-ketosteroid excretion in normal males from 17 to 40 years of age. The similar parallel fluctuation observed in this study tends to confirm such results and further emphasizes the sensitivity of the peptic cells to changes in circulating corticoids. (New England J. Med., Nov., 18, 1954; S. J. Gray, M. D., C. G. Ramsey, M. D., Peter Bent Brigham Hospital; R. W. Reifenshtein, M. D., Harvard Medical School, Boston)

* * * * *

Solitary Circumscribed Lesions of the Lung

The term "coin" lesion of the lung refers to a single, circumscribed, noncavitary lesion which causes no distal obstructive changes. This type of lesion is the most elusive from a diagnostic standpoint. It is essential to make an earnest effort to establish a correct diagnosis before considering surgical intervention. Unfortunately, this is fruitful in only about one-third of the cases. In the other two-thirds the lesion seems to defy all nonoperative diagnostic procedures. Bronchoscopy has been of little help except in those relatively rare instances where the lesions are centrally situated and involve a major bronchus. Similarly, bronchography discloses little more than that a lesion is present, a fact already determined by anteroposterior and lateral roentgenograms. Lymph node and supra-clavicular fat pad biopsies have value in the diagnosis of other types of lesions, but yield comparatively sterile results in "coin" lesions. Also, as pleural fluid is rarely associated with these lesions, thoracentesis is of no avail.

Certain tests, however, often give valuable clues to the real nature of the pathologic involvement. The most important procedure in the diagnostic work-up of these patients is the cytologic examination of the sputum for malignant cells. A preoperative diagnosis can be made in more than half of the cases of this type of bronchogenic carcinoma by this method, and this in spite of the fact that most of the lesions are situated in the periphery of the lung where collection of exfoliated cells is the most difficult. It is assumed that the laboratory is familiar with the proper collecting, fixing, and staining of the material, and this necessitates the development of a few special, though simple, skills. It also requires

considerable practice in viewing the slides to distinguish isolated malignant cells from regenerated and normal ones. However, as this is the only way short of bronchoscopic biopsy to make a preoperative tissue diagnosis, it is essential that this technic be perfected and used more widely.

The presence or absence of calcium in the lesion is of diagnostic import. In not a single instance in the Mayo Clinic series was malignancy present in a lesion containing calcium. This was true whether the calcium was punctate or laminated, and usually meant that the lesion was either a granuloma or a hamartoma. Most observers concur in this opinion, although it is often admittedly difficult to be certain that what is seen on the roentgenogram is definitely calcium.

When all diagnostic aids have been exhausted, two-thirds or more of these lesions remain a mystery. This is a mystery that deserves a rapid solution because one out of three will be cancer, either primary bronchogenic carcinoma, metastatic carcinoma, or sarcoma, bronchial adenoma, or primary connective tissue tumors. Exploratory thoracotomy with wedge resection, segmental resection, or even lobectomy, as a biopsy procedure is, therefore, mandatory. Whether the resection should then be extended to a lobectomy or pneumonectomy depends on the report from the frozen section. If the lesion is found to be benign, comparatively little lung tissue has been removed and a correct diagnosis can be established as a result of more thorough histologic and bacteriologic study. The physician also can be better guided as to subsequent medical management.

Solitary circumscribed lesions of the lung must be viewed with concern as one-third are malignant. All pertinent diagnostic procedures must be vigorously pursued, and the diagnostician should stop at nothing short of exploratory thoracotomy if the diagnosis remains obscure. Complacency, when dealing with such lesions, will usually lead to serious trouble. Thoracotomy should be performed without delay when the nature of the disease is indeterminate. Conservative pulmonary resection is done to establish the diagnosis so that definitive treatment can be undertaken. The risk of such a procedure roughly parallels that of laparotomy. (Postgrad. Med., Nov., 1954; R. W. Jamplis, Palo Alto Clinic, Palo Alto, Calif.)

* * * * *

Disappearing Bones

The gradual and often complete resorption of a bone or group of bones is an unusual phenomenon. It has been described under various titles such as phantom clavicle, acute absorption of bone, and disappearing bone. Occurring chiefly in young adults and frequently first discovered

after an injury--often a fracture--the slow dissolution ultimately results in complete disappearance of the affected bone or bones. Although the authors found 16 reported cases, some of them very well documented, few physicians have seen or heard of this uncommon form of progressive osteolysis. Moreover, in none of the various reports, has a satisfactory explanation of the pathogenesis of the lytic process been proposed and the etiology of the lesion is far from clear.

This report is based upon the personal observation of two cases of this unusual disorder, one with autopsy which permitted detailed study of the affected tissues, as well as a brief review of the 16 reported cases, some of which have been painstakingly followed up. As a result of this investigation it is now possible to record the clinical characteristics of a remarkable syndrome, that of massive osteolysis, and to give a description of the pathologic changes found in one case. The authors gave a possible explanation of the mechanism whereby the affected bones are slowly dissolved and reduced to little more than flexible fibrous bands.

On the basis of data from the authors' own cases and 16 others previously reported, the conclusion was reached that this unusual disease occurs generally in children or young adults. Only 4 of the patients were over 21 years of age; 10 were males, and 8 were females. In 15 cases there was a history of trauma, usually of minor character, at most associated with a fracture, and sometimes of pathologic type. However, unlike Sudek's Atrophy which develops soon after injury, a long interval had usually elapsed before the osteolytic process progressed far enough to give symptoms, and then the striking and unexpected roentgenographic findings have often come as a surprise. Thus the question arises whether trauma plays a definite etiologic role, or merely complicates a disease process already present, and in the end helps to bring it to light.

This unusual lytic disease is not limited to any one bone but may involve the clavicle, scapula, ribs, sternum, humerus, radius, ulna, jaw, bones of the hands or feet, femur, and pelvis. The slow disintegration affects both the cortical and cancellous elements of bone. It progresses slowly, and while the process may stabilize, it usually continues until practically all osseous tissue is gone, leaving only a fibrous band (presumably the periosteum) as a residuum.

The most striking pathologic feature in all cases in which biopsy specimens have been studied is a vascular abnormality of some kind, chiefly an overgrowth of small thin-walled vessels, a form of angiomatosis which in one instance was so striking that it suggested a malignant process. Generally, the vascular hyperplasia has been of blood vessel origin. Only one case was considered at first to be lymphatic but later reclassified as a hemangioma. None of the angiomatous changes, however, have been considered malignant and in nearly all cases the vessel overgrowth has not been neoplastic.

This unusual disease of bone progresses slowly and may cease to advance after a period of years. (Am. J. Med., Nov., 1954; L. W. Gorham, M. D., A. W. Wright, M. D., H. H. Shultz, M. D., and F. C. Maxon, Jr., M. D., Albany Medical College, Union University, Albany, N. Y.)

* * * * *

"Honors"

Two Navy medical officers were honored at the 61st Annual Meeting of the Association of Military Surgeons held in Washington, November 29-December 1, 1954.

Captain C. C. Shaw, MC USN, received the Association's Sir Henry Wellcome medal and prize for his essay on "The Clinical Syndrome of Acute Renal Insufficiency." This prize and medal was established by Sir Henry Wellcome in 1916. It is given annually by the Trustees of the Wellcome Foundation in London for the best essay on a military medical subject. The award consists of a silver medal, a scroll, and an honorarium of \$500.

Commander J. R. Seal, MC USN, was awarded the Edward Rhodes Stitt Award for his rigidly controlled studies of infections of the upper respiratory tract. These studies, conducted over a period of eight years, demonstrated that the oral administration of antibiotics provided an effective, inexpensive means of preventing or aborting epidemics of streptococcal infections.

Commander Seal is the first recipient of the Stitt Award. Presented annually, the award was established by Charles Pfizer and Company of Brooklyn, New York, to honor the memory of Rear Admiral E. R. Stitt, MC USN, who became the 16th Surgeon General of the Navy and made outstanding contributions to tropical medicine. The award, made annually to a member of the Association who has done outstanding work in the field of antibiotics, consists of a life membership in the Association, a metal plaque, and an honorarium of \$500. (TIO, BuMed)

* * * * *

Training Course in Special Weapons, Isotopes, and Military Medicine for Reserve Medical and Dental Officers

The fourth annual course, "Special Weapons, Isotopes and Military Medicine," will be sponsored by the Inspector, Naval Medical Activities, Pacific Coast, and presented by the Commandant, Twelfth Naval District, during the period 28 February - 4 March 1955, at the U. S. Naval Station, Treasure Island, San Francisco, Calif.

This course has been arranged to provide Reserve Medical Department officers of the Armed Forces the latest information to be employed in the many and varied aspects of special weapons, isotopes, and military medicine and dentistry. Each subject will be presented by a speaker of prominence in the specialty concerned.

Eligible Reserve officers will receive retirement point credits, on the basis of one (1) point for each day of attendance. Reserve Medical Department officers desiring point credits for attendance must obtain authority and appropriate orders to assure accreditation. Officers who hold appropriate duty orders, and a limited number of officers in the Active Status Pool, may be issued orders to active duty for training with pay. A tentative program and applications for active duty training and/or authorized orders will be mailed prior to 1 January 1955.

Naval Reserve Medical Department officers who have performed fourteen (14) days active duty for training, with or without pay; retired officers; or officers on the Inactive Status List are invited to attend this course without orders and will not receive retirement point accreditation.

Although this course is intended primarily for Naval Reserve Medical Department officers of the Pacific Coast, active duty personnel are invited to attend, as well as other components of the Armed Forces, the Public Health Service, and Civil Defense personnel. (DMO, 12th N. D.)

* * * * *

Notice of VD Postgraduate Course

The Twenty-Third Annual Venereal Disease Postgraduate Course will be given at Tulane Medical School of Louisiana in New Orleans, from January 31 through February 4, 1955. The course is being sponsored by the Division of Graduate Medicine at Tulane University in cooperation with the U.S. Public Health Service.

This course is designed to acquaint Medical Department officers, especially those working in venereal diseases, in the latest developments in diagnosis, treatment, and management of these diseases. As in the past, there will be no tuition charge for this course.

Applications for attendance or requests for information concerning the course should be made immediately to: Dr. Clifford Grulee, Jr., Director, Division of Graduate Medicine, Tulane University, 1420 Tulane Avenue, New Orleans, La.

These courses have been very well attended by naval officers in the past and it is hoped that their recommendations will encourage additional applicants this year.

Funds for travel and subsistence for personnel attending this course are not available from the Bureau of Medicine and Surgery. (PrevMed, BuMed)

Chemical Warfare Defense Training Film

Inquiries from field activities have been received by the Bureau of Medicine and Surgery concerning the advisability of baring the skin over the upper arm or thigh prior to the injection of atropine subsequent to exposure to the nerve gases under combat conditions. This method of procedure is depicted in the Navy training film MN-7396B, "Chemical Warfare Defense." When utilizing the training film, it should be pointed out that baring of the skin is desirable, provided there is little possibility of further exposure to chemical warfare agents while the skin is bared, and if an undue delay in administration of the atropine injection is not incurred as a result of baring the skin.

Whenever there is the possibility of further exposure, and particularly to agents in the liquid state, it is desirable to maintain the integrity of the protective clothing barrier. During a chemical warfare attack there is a strong possibility that drops of liquid type agents will be splashed or otherwise deposited on personnel. Although chemical warfare type of protective clothing will afford only slight protection against the percutaneous toxicity of the nerve gases in the liquid state, an initial measure of protection will be obtained in that the clothing absorbs a portion of the liquid before it penetrates to the skin. Thus, administration of atropine during an attack with the nerve gases should be through the clothing. To prevent percutaneous absorption of agent from the clothing it is essential to remove or cut away immediately the portion of the clothing that has been contaminated, after the attack is over.

Rapid injection of atropine is essential in a severely exposed person because the time between exposure and the resulting inability to self-administer atropine may be exceedingly limited. First-aid administration of atropine to a group of casualties will not allow time for baring of the skin. Exposure of the skin while wearing certain types of clothing, such as the Arctic clothing, would be very difficult as well as time consuming, and in cold weather might result in frostbite.

It is believed that the baring of the skin prior to atropine injection, as it is shown in the training film, could be interpreted in such a way that it would be unduly restrictive in its implications. As an adjunct to the training film, personnel responsible for training in chemical warfare defense should indicate the conditions under which baring of the skin is not desirable. (Audio-Visual Training Branch, BuMed)

* * * * *

The printing of this publication has been approved by the Director of the Bureau of the Budget, June 23, 1952.

* * * * *

The Navy Mutual Aid Association - Eligibility
of Reserve Officers for Membership

OPNAV NOTICE 1741, dated 24 September 1954, sets forth the conditions under which officers of the Navy, Marine Corps, and Coast Guard, including Reserve officers serving on active duty, may become members of the Navy Mutual Aid Association. The Navy Mutual Aid Association is a nonprofit, officer-controlled association established in 1879 under the auspices of the Secretary of the Navy for the purpose of providing immediate aid to the dependents of deceased officer personnel in the form of a substantial cash payment wired or cabled anywhere in the world, and in the prompt preparation and submission of all Government claims.

Those eligible for membership are: (a) Regular commissioned and warrant officers, both permanent and temporary, of the active lists of the Navy, Marine Corps and Coast Guard; (b) Reserve officers of these services serving on extended active duty, who have 1 year's continuous active service or 1 year or more of obligated service at time application is submitted. The maximum age for new members is 45 - 1/2 years.

Interested officers should address their inquiries to the Navy Mutual Aid Association, Navy Department, Washington 25, D. C.

* * * * *

From the Note Book

1. Rear Admiral B. W. Hogan, USN, Deputy and Assistant Chief of the Bureau of Medicine and Surgery, represented the Medical Department of the Navy at the launching of the USS Forrestal at the Newport News Shipyard on December 11, 1954. (TIO, BuMed)
2. On November 17, 1954, the Dental Department, Naval Station, Treasure Island, arranged a joint professional program for two Reserve Dental Companies and three local Medical-Dental study groups. The groups represented were: the California Academy of Periodontology, the 760 Club, the San Francisco Medical-Dental Study Club, and Dental Reserve Companies 12-1 and 12-2. (TIO, BuMed)
3. The Council on Dental Education, upon recommendation of the Council's Committee on Internships and Residencies, has approved the dental internship educational program being conducted at the Naval Hospitals, Corona, and Camp Pendleton, Calif. (TIO, BuMed)
4. A Navy selection board convened on November 30, 1954, to recommend Dental officers of the Regular Navy and Naval Reserve on active duty for promotion to the grade of lieutenant commander. The promotion zone

begins with LT Richard A. Dickson, Jr., and ends with LT Henry T. Mumme, Jr. (TIO, BuMed)

5. Dr. Mario Giaquinto, Chief of the Endemo-epidemic Disease Section of the World Health Organization, recently visited Naval Medical Research Unit No. 3 in Cairo, Egypt. In an informal discussion with the staff of NAMRU-3, he reviewed in some detail his recent experiences with sporadic rodent plague which occurs in certain areas of the Near East. (TIO, BuMed)

6. A radiation-balance microcalorimeter, recently developed by Dr. W.B. Mann of the National Bureau of Standards, precisely determines the emission rate of low-activity radioactive sources. The instrument does this by measuring the minute amounts of heat energy which accompany radioactive emission. The device is extremely compact and requires a relatively short time to complete a determination. It can be used to determine the intensity of a single source, or to compare two sources of nearly equivalent energy emission. (NBS, Summary Technical Report 1894)

7. Permissible Dose from External Sources of Ionizing Radiation, National Bureau of Standards Handbook 59, 79 pages, 30 cents. The recommendations and discussions of permissible dose contained in this Handbook form the basis of all other recommendations of the National Committee on Radiation Protection, including permissible doses for radio-active material within the body, safe handling of radioactive materials, waste disposal, et cetera.

The Handbook presents discussions of the basic concepts of permissible dose and discussions of each of the many factors considered in the formulation of the recommendations. For easy reference, the exposure limits of parts of the body to various types of ionizing radiation are briefly stated in the section, "Protection Rules." (NBS, Announcement, Oct., 11, 1954)

8. A series of 35 surgically resected discrete pulmonary granulomas, presenting typical macroscopic and microscopic features of the so-called tuberculoma, was subjected to an intensive search for acid-fast bacteria and fungi. Tubercle bacilli were found in Ziehl-Neelsen-stained sections of 6 lesions. Special stains for fungi demonstrated Coccidioides in 3 cases and Histoplasma in 19 cases. No organisms were found in 7 granulomas. (Arch. Int. Med., Nov., 1954; LtCol L.E. Zimmerman, MC USA)

9. The use of postmortem homografts to reduce mortality in extensive burns is reported in JAMA, 20 Nov., 1954; J. B. Brown, M.D., and M.P. Fryer, M.D.)

10. An Armed Forces standard field ration will replace the present B-type ration in the Navy and Marine Corps. With the new ration all three Services can utilize the same subsistence supplies and serve the same meals from kitchens where refrigeration is not available. (MSTS Bulletin, Dec., 1954)
11. Clinical features of a new environmental respiratory disease (Yokohama Asthma), occurring in certain areas of Japan during the winter months, are presented in Industrial Hygiene and Occupational Medicine, Nov., 1954; LtCol T.E. Huber, MC USA et al)
12. The interpretation of urologic symptoms in women is discussed in Postgrad. Med., Nov., 1954; L.R. Wharton, M.D.
13. A study of scleroderma, covering a period of approximately 14 years and including over 150 cases, is presented in Ann. Int. Med., Nov., 1954; I. Leinwand, M.D., A.W. Duryee, M.D., and M.A. Richter, M.D.
14. A review of the techniques commonly used in the management of jaw fractures in one large, active, municipal hospital, is presented in Arch. Otolaryng., Nov., 1954; R.N. Shapiro, M.D., D.D.S., G.R. O'Brien, M.D., and C. Willkie, D.D.S.
15. The current status of hypothermia is discussed in Arch. Surg., Nov., 1954; H. Swan, M.D.
16. The Northwest Airlines has received information from their District Official in Korea that quarantine officials are requiring persons arriving in Korea to be vaccinated within 6 months against typhoid and paratyphoid fever, typhus, and cholera. This is in addition to the smallpox vaccination of less than 3 years. (PHS, D.H.E.W.)

* * * * *

Board Certifications

American Board of Anesthesiology

LT Leo Scott M. Duflet (MC) USNR (Inactive)
LTJG Nicholas M. Greene (MC) USNR (Inactive)
LCDR Amedeo J. Losito (MC) USNR (Inactive)

American Board of Internal Medicine

LTJG Charles A. Bailey (MC) USNR (Inactive)

LT John V. Dean (MC) USNR (Inactive)
LT Rowland D. Goodman II (MC) USNR (Inactive)
LTJG David L. Hoffman (MC) USNR (Inactive)
LT Dan C. Roehm (MC) USNR (Inactive)

American Board of Obstetrics and Gynecology

LT Bernard M. Altschuler (MC) USNR (Inactive)
LT William E. Byrd (MC) USNR (Inactive)
LTJG Edmund Furcinto (MC) USNR (Inactive)
LCDR Irving Greene (MC) USNR (Inactive)

American Board of Ophthalmology

LT Arnold W. Forrest (MC) USNR (Inactive)
LT John P. Luhr (MC) USNR (Inactive)
LTJG Frank C. Winter (MC) USNR (Inactive)

American Board of Orthopedic Surgery

LT Richard M. Fitzsimons (MC) USNR (Inactive)

American Board of Otolaryngology

LT Jesse D. Gomillion, Jr. (MC) USNR (Inactive)

American Board of Pathology

LT Theodore C. Keller (MC) USNR (Inactive)

American Board of Pediatrics

LT Forest A. Cornwell (MC) USNR (Inactive)
LT Samuel C. Dunn (MC) USNR (Inactive)
LT Francis L. Jaubert, Jr. (MC) USNR (Inactive)

American Board of Psychiatry and Neurology

LT Marvin G. Drellich (MC) USNR (Inactive)
LT Ernest Gosline (MC) USNR (Inactive)
LCDR Joseph D. McElroy (MC) USNR (Inactive)

American Board of Radiology

LT James L. Krieger (MC) USNR (Inactive)
LTJG Harvey J. Thompson, Jr. (MC) USNR (Inactive)

American Board of Surgery

LCDR Edwin C. Bebb (MC) USNR (Inactive)
LT Elmer B. Campbell, Jr. (MC) USNR (Inactive)
LTJG Howard S. Jeck (MC) USNR (Inactive)
LT Matthew A. Larkin (MC) USNR (Inactive)

CDR John J. Lille (MC) USNR (Inactive)
LTJG John B. White, Jr. (MC) USNR (Inactive)

American Board of Urology

LTJG John T. Hicks, Jr. (MC) USNR (Inactive)
LTJG Roland B. Laury (MC) USNR (Inactive)

American College of Surgeons

CAPT E. F. Norwood (MC) USN
LCDR M. J. Albers (MC) USNR (Inactive)
LCDR J. E. Brennan (MC) USNR (Inactive)
CDR J. Crawford (MC) USN
CAPT Shakeeb Ede (MC) USN

* * * * *

BUMED NOTICE 6710

26 November 1954

From: Chief, Bureau of Medicine and Surgery
To: Distribution List

Subj: Narcotic drugs; open purchase of

Ref: (a) BUMEDINST 6710.6

This Notice promulgates a change to reference (a).

* * * * *

BUMED NOTICE 6630

3 December 1954

From: Chief, Bureau of Medicine and Surgery
To: All Ships and Stations Having Dental Corps Personnel
Regularly Assigned

Subj: Submission of NavMed Forms L

This Notice advises that the subject forms are frequently mutilated and sometimes lost in transit to the Bureau of Medicine and Surgery as a result of careless packaging.

* * * * *

BUMED NOTICE 6320

8 December 1954

From: Chief, Bureau of Medicine and Surgery
To: All Ships and Stations Having Medical Personnel
Regularly Assigned

Subj: Report on Number of Outpatient Visits, Medical Treatment
Facilities for January, 1955

In order to provide additional information for use in connection with pending legislation on medical care for dependents, the Department of Defense has requested that the number of outpatient visits at naval medical treatment facilities during January 1955 be reported.

* * * * *

Change of Address

Please forward requests for change of address for the News Letter to: Commanding Officer, U.S. Naval Medical School, National Naval Medical Center, Bethesda 14, Md., giving full name, rank, corps, and old and new addresses.

* * * * *

AVIATION MEDICINE DIVISION

Handling Casualties Aboard the USS Bennington

The last AirLant Safety Council meeting was an interesting one. Experiences recalled from the USS Bennington explosion are enough to make each one of us wonder, "How would my unit perform under the same conditions?" Both Captain Kirk Smith, ashore at Quonset Point, and

Lieutenant Ledwith, on board the USS Bennington, were modest in regard to the superb job done by all medical officers and hospital corpsmen concerned. They both purposely tried to confine their presentation to the things that they thought could be improved. The general ideas which they presented are as follows:

1. Lives were saved by non-medical personnel who knew their bleeding pressure points, but there is need for more drilling in life-saving first-aid procedures. The use of a Moulage Set (Stock No. 6910-786-050, Unit price \$24.16) could perhaps condition timid personnel to the appearance of blood and gore. Confidence in ability attained in drills will probably be the best way to reduce the ranks of squeamish non-medical personnel. There was reason to postulate that fatal bleeding took place in some injured that might have been stopped if the non-medical personnel who reached them first were more confident in their ability to help.

2. When disaster of great proportion occurs on a carrier, it would be wise to make up a collecting station on the hangar deck as soon as possible. Large numbers of casualties in passages approaching the sick bay clog the passage. If the casualties are put in an open space, a trained eye could spot the urgent cases faster.

3. Dextran (plasma expander) is an excellent electrolyte restorative and probably will completely cover the needs of immediate shock treatment on carriers and other operating units. There is no substitute for blood when needed, but in most burn cases, electrolyte is the immediate need. Transfer of patients before blood is indicated can normally be expected.

4. Be unrelenting in the campaign to get all people in operating units to wear their identification tags. Remind them of the anguish to their family by lack of identification and of the loss of valuable time and personnel caused by carrying on a "Hawkshaw" detective routine during a major catastrophe. Note: Recently a plane went down--all seven occupants were lost. All of the bodies were finally recovered and none were wearing their identification tags.

5. Help in these modern times comes sooner than you think. Dr. Ledwith said he and Dr. Norman found helicopters aboard available for patient evacuation before they had time to figure out who should go out first. The doctors were busy. Probably one corpsman should be assigned to this task.

6. Tag articles of sentimental or actual value as soon as they are removed from a person.

7. Have one person as an information center. That one might make out a small library card for each casualty showing name, location, and condition. If that casualty is moved from the hangar deck to the sick bay or from the hangar deck to a helicopter going ashore, the information should go to that one person in writing if possible. This would provide

a running alphabetical account which is invaluable. This service could be performed by non-medical personnel. (Air Force U.S. Atlantic Fleet, Medical News Letter, 23 September 1954)

* * * * *

Carbon Monoxide Blood Determination

Of interest to all medical officers on duty within the continental limits of the United States is BuMed Instruction 6510.4, 30 September 1954. This instruction deals with the preparation and shipping of blood specimens obtained from aviators and aircrewmembers for carbon monoxide determination and is of particular interest to flight surgeons. However, it is not restricted to them, because, in many aircraft accidents or near accidents, the pilot and other aircrew members are first seen by the duty medical officer of an infirmary or perhaps a hospital who in many cases will not be a flight surgeon.

To improve the medical investigation of the physiological causes of aircraft accidents or near accidents, it is desired that all cases of suspected cockpit contamination be thoroughly investigated. Where carbon monoxide poisoning is a reasonable possibility, submission of a blood sample to the Department of Toxicology for Carbon Monoxide Determination, U.S. Naval Medical School, National Naval Medical Center, Bethesda 14, Maryland, in accordance with the detailed instructions promulgated by BuMed Instruction 6510.4, is indicated.

The use of local naval medical laboratories or other local laboratory facilities is to be encouraged, and every effort should be made to develop as complete an investigation as possible with the facilities available. In all cases where it is possible to obtain more than the minimum amount of blood necessary for the carbon monoxide determination test, in compliance with this new instruction, the medical officer is encouraged to submit a sample of blood to the locally available laboratory. (Please note that this is in addition to the specimen submitted to the U.S. Naval School of Medicine, National Naval Medical Center, Bethesda 14, Maryland.) The completion and forwarding of the Medical Officers Report of Aircraft Accident should not be delayed for the purpose of including the report from the Naval Medical School laboratory. Any local reports should be included if possible.

Unconsciousness of pilots from unknown causes is still taking its toll of lives and medical officers should do their utmost to solve this challenging problem.

* * * * *

The Pressure Demand Oxygen Mask

Question: When difficulty is experienced exhaling from a pressure demand oxygen mask, the probable source of trouble is (a) the exhalation valve, or (b) an inlet valve?

Recently, this question was posed to a group of ten experienced pilots on a short oxygen equipment quiz. Only one of the ten knew the correct answer. He alone knew how to correct such a difficulty.

The right answer to the question is (b). The exhalation difficulties with the pressure mask are in the inlet valves.

Several factors may prevent a positive inlet valve seal: (a) If the rubber portion of the valve is warped, or if dirt is caught underneath, it won't seal properly. (b) If the plastic base for the valve is not completely inserted in its receptacle, there can be no seal.

On some small and medium masks, the microphone port may be pressed against the inlet valve structure and may cause a leak between the rubber portion of the mask and the plastic base for the inlet valve.

Of the two preceding factors, the first is the most common, and keeping the mask and particularly the inlet valves clean, will eliminate most exhalation difficulties. Should an inlet valve become warped, it should be replaced. Care must be taken to check for proper insertion of the inlet valve into the mask.

In the third instance, however, the source of trouble may easily escape detection. Binding between the microphone port and the inlet valve has occurred primarily on small masks and occasionally on medium sizes. The remedy is to insert an additional rubber gasket around the plastic base of the inlet valve so that a tighter fit is provided between the mask proper and the inlet valve base.

It can be seen that if one of the inlet valves leaks or does not seal properly on exhalation, the force of exhalation is not only exerted on top of the exhalation valve, but is vented back through the inlet valve into the mask oxygen hose and then through the exhalation valve extension up to the underside of the exhalation valve. Consequently, with equal pressures present on both the top and bottom of this valve, the springs operate to keep the valve shut, and exhalation must take place by blowing the mask loose from the face.

Take the time to disassemble your mask and see how it works. When you have trouble exhaling, you should then have little difficulty in locating the source of trouble. (USAF Flying Safety Magazine, Apr., 1954)

* * * * *

Instructor's High Altitude Physiology Training Manual

A new U. S. Navy Instructor's High Altitude Physiology Training Manual, NAVEXOS P-1260, has recently been published by the U. S. Navy Special Devices Center at Port Washington, Long Island, New York, for use by flight surgeons, aviation physiology training officers, and aviation technicians concerned with training aviators and aircrewmen in low pressure chambers.

This manual is the result of the combined efforts of medical department officers in the field, the Aviation Medicine Division of the Bureau of Medicine and Surgery, and personnel at the Special Devices Center. It is hoped that it will be of assistance to those who are charged with the responsibility of training aviation personnel in the use of airborne oxygen equipment and the phenomena experienced at high altitudes.

It is also hoped that this manual will, to some extent, standardize the low pressure chamber programs throughout the Naval Establishment. Material for lectures pertaining to atmospheric physics, anatomical and physiological factors, equipment and procedures, as well as duties and responsibilities of the chamber personnel are all detailed. Model questionnaires, suggested safety measures and precautions, and a bibliography of pertinent publications are included to complete this new manual.

Undoubtedly, this manual will contain controversial points, and possibly a few errors; however, on the whole, it should prove a helpful bit of guidance to most of those charged with the responsibilities of teaching aviation physiology and the operation of the low pressure chambers..

* * * * *

Pressure Breathing Training

In order to somewhat save "wear and tear" on our low pressure chamber inside observers, and still adequately train aviators and aircrewmen in the use of the automatic diluter-demand pressure breathing oxygen regulator and equipment, it has been decided to adjust "P" series 2867 regulator models so as to deliver oxygen to the user at a pressure of approximately 10 inches of water at a simulated pressure altitude of 35,000 feet.

Thus, the regulator mechanism so adjusted will automatically cut in pressure breathing at about 28,500 feet simulated altitude and progressively increase to 10.6 inches of water pressure at 35,000 feet.

Letters establishing the procedure for the adjustment of these regulators have been received by senior medical officers of stations at which low pressure chambers are located. This letter states that the procedure was applicable to Models 2862 and 2867. This is in error, as the procedure

outlined is applicable only to regulators with a "P" designation in the serial number, for example, Serial Number M 1359P.

These regulators and a few others that have been modified have a button instead of a phosphor bronz leaf spring on the lever mechanism which activates the demand valve when the pressure breathing aneroid expands.

The second factor in being able to set the regulator for lower altitude pressure breathing is the characteristic of each individual aneroid. By testing, it has been found that the aneroid must have an expansion of at least .01 inches when the ambient atmospheric pressure is reduced from sea level to 28,400 feet in order for this adjustment to be made. Approximately two out of three aneroids will meet this requirement. Therefore, in order to successfully adjust the regulators as previously recommended, it is necessary to, first, use the "P" series of Model 2867 regulators and, secondly, following the previously recommended adjustment instructions, check each regulator for sea level leakage and, lastly, check for altitude "cut in" accuracy by the use of a simple water manometer in a low pressure chamber and make corrective adjustments as needed. Bear in mind the fact that approximately one out of every three will not be usable because of the characteristics of the aneroid.

It behooves all flight surgeons, aviation physiology training officers, and aviation technicians to read and know this procedure and to understand the ground rules for tagging the regulators and entering notations of training in the subject's health record.

* * * * *

Founders Group of Aviation Medicine Certification

The American Board of Preventive Medicine has completed its selection of physicians to be certified in aviation medicine and known as the Founders Group. There were 170 chosen of which 36 are United States Navy; 4, United States Navy, Retired; 4, United States Naval Reserve, Inactive; and 1, United States Naval Reserve, Retired. The following listed officers were so honored:

United States Navy (36)

Captain Norman L. Barr
Captain Thenton D. Boaz
Commander Sidney I. Brody
Captain Elmer L. Caveny
Captain Oran W. Chenault
Commander Ralph L. Christy

Rear Admiral Winfred P. Dana
Commander Calvin T. Doudna
Captain Julius C. Early
Captain Thomas Ferwerda
Captain David C. Gaede
Commander Perry W. Gard

Captain Charles F. Gell	Captain Herbert G. Shepler
Captain Merrill H. Goodwin	Commander Harold A. Smedal
Captain Ashton Graybiel	Captain F. Kirk Smith
Captain John H. Korb	Captain John T. Smith
Captain Arthur W. Loy	Commander Frank B. Voris
Captain Lester E. McDonald	Captain Raphael L. Weir
Captain Earle E. Metcalfe	Captain James L. Holland
Captain Langdon C. Newman	Commander Walton L. Jones
Commander Philip B. Phillips	Captain Wilbur E. Kellum
Captain Clifford P. Phoebus	Captain Samuel J. Wisler
Commander Joseph P. Pollard	Captain Jesse G. Wright
Commander Kenneth S. Scott	Commander Edward M. Wurzel

United States Navy, Retired (4)

Captain Leon D. Carson	Captain Louis E. Mueller
Rear Adm. Bertram Groesbeck, Jr.	Captain John R. Poppen

United States Naval Reserve, Inactive Status (4)

Commander Marion M. Kalez	Captain Bernhard R. Reinertsen
Captain Lawrence M. Larson	Captain Herman D. Scarney

United States Naval Reserve, Retired (1)

Commander Henry A. Schroeder

* * * * *

Zurich, Switzerland, 1954

The French-speaking Branch of the Aero Medical Association held its third annual meeting in Zurich, Switzerland, 20-23 September 1954. Major K. Wiesinger, Chief Flight Surgeon of the Swiss Air Force and President of the Branch, presided. The meeting was an unqualified success with more than 100 flight surgeons from 14 widely scattered countries attending.

The scientific portion of the meeting was held in the new Cantonal Hospital in Zurich and was fully attended during each session. Facilities for professional and social events of the meeting were arranged under the patronage of Colonel Brigadier H. Meuli, Surgeon General of the Swiss Armed Forces; Dr. P. Meierhans, President of the Zurich State Council; Dr. E. Landolt, Mayor of Zurich; Dr. W. Spuehler, Counselor of Zurich; Dr. W. Berchtold, Chairman of the Board of Directors of Swissair;

Dr. M. Burkhard, Chairman of the Federal Office of Civil Aviation; and Dr. W. Muri, President of the Aero Club of Switzerland.

The opening address of welcome was delivered by Colonel Divisionnaire E. Primault, Chief of the Swiss Air Force. The scientific program included presentations in main topics such as, the regulation of medications among flying personnel, the psychological aspects of military aviation, and the role of the circulatory system in commercial pilots.

The social program included luncheons at three of Zurich's most colorful and famous restaurants and visits to the new Kloten Airport, Zurich, and the Aero Medical Institute of the Swiss Air Force located at Dubendorf. A cocktail reception followed these visits. The former was sponsored by the Canton of Zurich, and the latter by the Aero Club of Switzerland. The annual banquet was the closing activity at Zurich and was held at the city's most famous historic guild house, the Haus zum Ruden. Addresses were given by Colonel Brigadier H. Meuli, Medicine-General Pierre Bergeret, and Major General Harry Armstrong. General Armstrong, representing Brigadier General Otis Benson, President of the Aero Medical Association, expressed General Benson's regrets for not being able to attend the meeting. During the business meeting, General Bergeret was elected president of the Branch for the coming year, and he announced that the next year's meeting would be held somewhere in France, the definite site to be announced later. Following the formal program, trips of one day each were scheduled to Mount Rigi, near Lucerne, and to Interlaken for an over-night stay to be followed by a visit to the International Physiological Institute atop Jungfrauoch and thence to Bern, returning to Zurich the second night.

Many medical directors of United States and Canadian airlines were present, as well as representatives of the Airline Medical Examiners Association, Airline Medical Directors Association, the Bureau of Medicine and Surgery, U.S. Navy, and the Surgeon General's Office of the U.S. Air Force. More than a score of American and Canadian officers on duty in Europe attended; many were accompanied by their wives.

A social program for the wives of delegates was most interesting and inclusive of all points of interest in and near Zurich. This kept the ladies happily busy during the scientific session.

* * * * *

Defects Noted on SF-88's Submitted to BuMed:
October and November 1954

Excess copies	201
Lack of copies	25
Copies not legible	5

Original and copies dissimilar	3
Item 1. (Name incomplete or in error).....	41
Item 2. (No designator here or elsewhere)	87
Item 5. (Purpose of examination omitted)	1
Item 6. (Date of examination omitted).....	7
Item 11. (Organizational unit omitted)	1
Item 12. (Birth date omitted or in error)	54
Item 15. (Examining facility omitted)	7
Item 17. (Aviators flight time omitted).....	84
Item 44. (No reason given for dental disqualification).....	1
Item 45. (Urinalysis omitted)	26
Item 45. (Specific gravity omitted)	49
Item 46. (Chest x-ray omitted)	11
Item 51. (Obvious errors in height)	8
Item 52. (Weight omitted or in error).....	5
Item 57. (Blood pressure omitted).....	9
Item 57. (C. E. R. omitted).....	18
Item 58. (Pulse omitted)	2
Item 59. (Distant vision omitted)	4
Item 60. (Refraction not properly recorded)	19
Item 60. (Refraction omitted on NavCad applicants)	11

* * * * *

☆ U. S. GOVERNMENT PRINTING OFFICE: 1954 O—311214

PENALTY FOR PRIVATE USE TO AVOID
PAYMENT OF POSTAGE, \$300

DEPARTMENT OF THE NAVY
BUREAU OF MEDICINE AND SURGERY
WASHINGTON 25, D. C.
OFFICIAL BUSINESS
Permit No. 1048